HRCT in uncommon occupational lung diseases - Do we need some help?

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Learning objectives

To review the diagnostic steps of some uncommon occupational lung diseases (OLD), some of them found in the PACS systems of two tertiary health care institutions - Universitair Ziekenhuis Brussel (Brussels, Belgium) and Hospital of Lithuanian University of Health Sciences Kauno Klinikos (Kaunas, Lithuania).

Background

Occupational lung diseases are among the most frequently diagnosed work-related conditions. OLD comprise of various disorders secondary to the inhalation of dust particles or noxious chemicals by workers in certain specific occupation. They include pneumoconiosis, asbestos-related pleural and parenchymal disease, chemical pneumonitis, infection, hypersensitivity pneumonitis, and organic dust toxic syndrome.

HRCT features of silicosis, asbestosis and coal-worker's pneumoconiosis have been widely described and are well known, unlike some more rare occupational lung diseases such as acute silicosis (silicoproteinosis), siderosis, talcosis, hard metal pneumoconiosis (giant cell interstitial pneumonitis) and chemical pneumonitis.

Findings and procedure details

Imaging plays a crucial role in the evaluation of occupational lung disease. Radiograph of the chest has a well-established role and can be highly suggestive of an occupational disorder, and sometimes, with an appropriate exposure history, may even be sufficient for making a diagnosis. More often findings on the chest radiograph are nonspecific and the sensitivity is low.

It has been proved beyond question that CT, particularly high-resolution CT (HRCT), is superior to chest radiography in the detection of parenchymal abnormalities, and is more accurate in providing differential diagnosis.

Uncommon OLD may mimic various other lung pathologies on HRCT because of the lung's quite limited capacity of response to injury, regardless of the cause. The features, which we see on HRCT, often are non-specific and appear as centrilobular nodules, patchy or diffuse ground-glass opacities, reticular opacities, architectural distortion, traction bronchiectasis- bronchiolectasis or honeycombing.

Diagnosis of an occupational lung disease requires:
• Definite history of exposure to an agent known to cause interstitial lung disease (ILD),
• An appropriate latency period,
• A consistent clinical presentation,
• Physiologic and radiologic pattern,
• Exclusion of other known causes of ILD.

In the presence of a history of exposure and consistent clinical features, the diagnosis
of even an uncommon occupational lung disease can be suggested by the described
characteristic HRCT findings.

We try to explain the clinical and radiological presentation of acute silicosis
(silicoproteinosis), siderosis, talcosis, giant cell interstitial pneumonitis and chemical
pneumonitis with the help of the pathophysiology of the lung and exposure history.

**Hard metal pneumoconiosis**

Hard metal pneumoconiosis, formerly classified as giant cell interstitial pneumonia,
results from exposure to tungsten carbide, cobalt and diamond dust produced in hard-
metal industry.

Hard metal pneumoconiosis is a rarely investigated disease, so the clinicopathological
features, HRCT findings, treatment and prognosis have not been clearly defined. It
consists of a spectrum of diseases containing occupational asthma and obliterative
bronchiolitis, as the earliest manifestations, and giant cell interstitial pneumonia and
interstitial fibrosis in the late course of disease.

Most patients have poor outcome. A decline in pulmonary function is observed despite the
steroid treatment and cessation of exposure. Occasionally pulmonary fibrosis develops.

The pathologic features of hard metal pneumoconiosis include thickening of interstitium
and alveolar walls, caused by mononuclear cell infiltration, filling of the air spaces with a
large number of macrophages and multinucleated giant cells.

The radiographic findings are nonspecific. The chest radiograph may be normal or show
a nodular, reticulonodular, or reticular pattern, patchy irregular opacities in middle and
lower lung zones.

HRCT findings of hard metal pneumoconiosis *(Fig. 1 on page 6 , Fig. 2 on page
7)*:
• Bilateral patchy lobular ground glass opacities,
• Tiny centrilobular nodules,
• Consolidation,
• Linear opacities and reticulation,
• Subpleural bullae,
• Long-term exposure: fibrosis with architectural distortion, traction bronchiectasis, honeycombing,
• Middle and lower zone predominance of above mentioned features.

GIP shares many of the clinical and other features of interstitial pneumonias, therefore a proper occupational history is essential for the diagnosis.

**Acute Silicosis**

Acute silicosis (or silicoproteinosis) occurs after a very large, acute exposure to silica dust, primarily among sandblasters. Usually more than 20 years of exposure to silica dust is needed to develop simple silicosis, while acute silicosis can develop after a period ranging from few weeks to several years of exposure to large amounts of silica dust.

Crystalline silica particles cause direct injury to alveolar walls. Pathologic features of silicoproteinosis, including alveolar filling with phospholipids and proteins, resemble those of alveolar proteinosis and differ substantially from those of chronic silicosis.

The rapid course of the disease usually leads to progressive respiratory impairment. Most reported cases have been fatal within months or few years.

Radiographic findings, like that of primary alveolar proteinosis, are bilateral parenchymal consolidations. Central ”butterfly” alveolar opacities with air bronchograms and hilar/mediastinal lymphadenopathy are common.

HRCT findings of acute silicosis (Fig. 3 on page 8, Fig. 4 on page 9):
• Multiple bilateral centrilobular nodules,
• Multifocal patchy ground-glass opacities,
• Bilateral consolidation in the posterior portions of the lungs with occasional crazy paving (calcification within areas of consolidation is a common finding),
• Hilar lymphadenopathy (with or without punctate or eggshell type calcifications),
• Later on the course of the disease - evolution to fibrosis with severe architectural distortion, bullae, pneumothorax.

**Talcosis**

Talc is hydrated magnesium silicate. Inhalation of talc can occur during its extraction from mines, separation, milling, packing, it is also used in leather, ceramic, paper, plastics, rubber, building, paint, and cosmetic industries.
The term "talc" is used not only for pure magnesium silicate but often for mixtures with various other products also, such as silica or asbestos. Inhalation of such products can result in clinical and radiological appearance more resembling of silicosis or asbestosis.

Even a relatively short but high exposure to talc may be a significant factor in the development of disease.

Symptoms are non-specific, including cough and chronic dyspnea progressing to cor pulmonale in end-stage disease.

Inhaled talc particles cause a nonnecrotizing granulomatous inflammation, resulting in diffuse micronodular pattern with well-defined nodules on chest radiograph in earlier stages of disease. In long-standing disease the nodules tend to confluence, producing large foci of consolidation associated with progressive fibrosis.

HRCT findings of talcosis (Fig. 5 on page 10):
• Multiple small centrilobular and subpleural nodules (that may calcify), predominantly in upper lung zones,
• Aggregation of nodules into heterogeneous conglomerate masses (identical to silicosis) that may have internal foci of high attenuation that correspond to talc deposition,
• May evolve into progressive massive fibrosis (PMF), with adjacent architectural distortion,
• (Pleural thickening, pleural and diaphragmatic plaques identical to asbestos-related pleural disease),
• (Enlarged hilar lymph nodes with eggshell calcification (especially in silico-talcosis)).

Siderosis

The majority cases of siderosis are seen in electric-arc and oxyacetylene welders. With the heat from the arc or torch, arc welding melts and boils the iron, and emitted ferric oxide appears as blue-gray fumes. Most of the particles present in these fumes are of respirable size. Other occupations at risk include mining and processing of iron ores, iron and steel rolling mills, foundry workers and silver polishers.

It has to be noted, that pulmonary hemosiderosis, which has a similar appearance, can develop due to various causes of recurrent alveolar hemorrhage, main of the reasons being mitral valve stenosis.

Siderosis is not usually associated with fibrosis or functional impairment. The radiologic abnormalities are reversible and may resolve partially or completely after exposure ceases, although symptomatic disease and interstitial fibrosis has been described in arc welders.
Siderosis is caused by the accumulation of iron oxide in macrophages within the lung. The micronodules on CT correspond to dust macules, which are collections of dust-laden macrophages aggregated along the perivascular and peribronchial lymphatic vessels.

On chest radiograph small rounded opacities widely distributed throughout both lung fields can be seen.

HRCT findings of siderosis (Fig. 6 on page 11, Fig. 7 on page 12):
- Widespread ill-defined small centrilobular nodules,
- Less commonly, patchy areas of ground-glass attenuation without zonal predominance,
- Emphysema,
- Findings of interstitial fibrosis, septal thickening with or without honeycombing.

Chemical pneumonitis and acute respiratory distress syndrome due to metal fumes from welding have also been reported.

**Chemical Pneumonitis**

The inhalation of noxious chemical substances, though not common, is a significant cause of occupational lung disease. These chemicals include organic (organophosphates, smoke, polyvinyl chloride, polymer fumes), inorganic (ammonia, hydrogen sulfide, nitrogen oxide, zinc chloride), and metal (cadmium, mercury, nickel).

Lung injury after exposure is primarily attributed to the inhalation of noxious chemical substances, which can rapidly cause respiratory mucosal damage.

The pathologic changes include pulmonary edema, pneumonitis, alveolar collapse, diffuse alveolar damage, and pulmonary fibrosis

HRCT findings of chemical pneumonitis (Fig. 8 on page 12):
- In acute exposure:
  o Centrilobular or patchy areas of ground-glass opacity, presumably due to pulmonary edema.
- Weeks to months after the exposure:
  o Bronchiolitis obliterans may develop, with findings of bronchiectasis, bronchiolectasis, mosaic perfusion, and air trapping.

**Images for this section:**
Fig. 1: Hard metal (nickel) pneumoconiosis in a 52-year-old lathe machine worker. Axial HRCT image shows minimal bilateral central cylindrical bronchiectasis, fine nodules, and mosaic attenuation, suggestive of obliterative bronchiolitis

Fig. 2: A 38-year-old machinist with hard metal pneumoconiosis. Axial HRCT image shows numerous poorly defined centrilobular nodules in the upper lobes.

Fig. 3: HRCT scan, coronal reformation, showing bilateral multiple small centrilobular nodules, interlobular septal thickening and patchy ground glass opacities, predominantly in upper parts of the lungs in a young worker with acute silicosis.

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**Fig. 4:** HRCT scan, coronal reformation in mediastinal window, showing mediastinal and bilateral hilar lymphadenophaty in a young worker with acute silicosis.

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Fig. 5: Talcosis in a 63-year-old woman working in rubber industry. Axial HRCT images show small centrilobular nodules (black arrows, A) in upper lobes. In the mediastinal windows (B, C), conglomerate mass is seen to contain high-attenuation material (curved arrow); it is also seen in mediastinal lymph nodes (arrows, C)

**Fig. 6:** Chest radiograph (A) demonstrating diffuse generalized reticular nodular shadowing and a chest CT scan (B) showing bilateral tiny nodular opacities throughout both lung fields predominantly in the mid and upper zones in a patient with siderosis.


**Fig. 7:** Siderosis with interstitial fibrosis in a lathe machine worker. Axial HRCT images show interlobular and intralobular septal thickening with honeycombing (arrow) in upper lobes

**Fig. 8:** Axial HRCT images showing bilateral patchy ground glass opacities, presumably due to pulmonary edema, in a patient with toxic pneumonitis, which was caused by zinc chloride inhalation.

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Conclusion

Occupational lung disease is a diverse group of preventable pulmonary diseases. Recognition of occupational lung disease is important not only for the primary worker, but also for exposed co-workers, with regard to primary and secondary disease prevention.

HRCT has an important role in diagnosis and assessment of activity of the diffuse lung diseases, including the ones caused by specific occupational exposure.

The characteristic radiological features suggest the correct diagnosis in some, whereas a combination of clinical features, occupational history, and radiological findings is essential in establishing the diagnosis in others.

In uncommon occupational lung disease only the combination of an appropriate exposure history, an appropriate latency period and a consistent clinical and radiological presentation can establish a correct diagnosis and avoid unnecessary radiation exposure or biopsies.

Personal information

References


